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Abstract

Background Physical therapists may prescribe stretching exercises for individuals with stroke to improve joint integrity and to reduce the risk of secondary musculoskeletal impairment. While deficits in passive range of motion (PROM) exist in stroke survivors with severe hemiparesis and spasticity, the extent to which impaired lower extremity PROM occurs in community-ambulating stroke survivors remains unclear. This study compared lower extremity PROM in able-bodied individuals and independent community-ambulatory stroke survivors with residual stroke-related neuromuscular impairments. Our hypothesis was that the stroke group would show decreased lower extremity PROM in the paretic but not the nonparetic side and that decreased PROM would be associated with increased muscle stiffness and decreased muscle length.

Methods Individuals with chronic poststroke hemiparesis who reported the ability to ambulate independently in the community (n = 17) and age-

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matched control subjects (n = 15) participated. PROM during slow (5 degrees/sec) hip extension, hip flexion, and ankle dorsiflexion was examined bilaterally using a dynamometer that measured joint position and torque. The maximum angular position of the joint (ANG_{max}), torque required to achieve ANG_{max} (T_{max}), and mean joint stiffness (K) were measured. Comparisons were made between able-bodied and paretic and able-bodied and nonparetic limbs.

Results Contrary to our expectations, between-group differences in ANG_{max} were observed only during hip extension in which ANG_{max} was greater bilaterally in people post-stroke compared to control subjects ($P \leq 0.05$; stroke = 13 degrees, able-bodied = −1 degree). T_{max}, but not K, was also significantly higher during passive hip extension in paretic and nonparetic limbs compared to control limbs ($P \leq 0.05$; stroke = 40 Nm, able-bodied = 29 Nm). Compared to the control group, T_{max} was increased during hip flexion in the paretic and nonparetic limbs of post-stroke subjects ($P \leq 0.05$, stroke = 25 Nm, able-bodied = 18 Nm). K in the nonparetic leg was also increased during hip flexion ($P \leq 0.05$, nonparetic = 0.52 Nm/degree, able-bodied = 0.37 Nm/degree.)

Conclusion This study demonstrates that community-ambulating stroke survivors with residual neuromuscular impairments do not have decreased lower extremity PROM caused by increased muscle stiffness or decreased muscle length. In fact, the population of stroke survivors examined here appears to have more hip extension PROM than age-matched able-bodied individuals. The clinical implications of these data are important and suggest that lower extremity PROM may not interfere with mobility in community-ambulating stroke survivors. Hence, physical therapists may choose to recommend activities other than stretching exercises for stroke survivors who are or will become independent community ambulators.

Keywords: cerebral vascular accident (CVA), hemiparesis, muscle, range of motion (ROM), spasticity

Introduction

Decreased passive range of motion (PROM) of joints is a common musculoskeletal problem for individuals with chronic poststroke hemiparesis.[1–6] Stroke survivors with severe hemiparesis and spasticity can develop joint contractures that cause limb deformities, pressure ulcers, and mobility problems.[2,3,5–7] Consequently, physical therapists may recommend stretching

exercises for people with stroke to improve joint integrity and to reduce the risk of secondary musculoskeletal impairment.[8,9] However, it is unclear whether less severely impaired individuals, particularly stroke survivors who regain the ability to ambulate independently in the community, have PROM deficits. Determining whether lower extremity PROM is decreased in community-ambulating stroke survivors is important because, if present, these deficits may contribute to locomotor dysfunction[7,10] and should be corrected or prevented with appropriate treatment. Alternatively, if PROM is adequately maintained in this subpopulation of stroke survivors, it may be advantageous for physical therapists to focus on other rehabilitation activities that may have a more substantial impact on recovery of function.

Little is known about the changes in lower extremity PROM that are associated with successful hemiparetic gait. The orthopedic literature contains numerous descriptions of joint deformities post-stroke. The most common of these impairments is the equinus or equinovarus deformity of the ankle[2,3,6,7]; however, hip, knee, and toe flexion contractures have also been reported.[2,3,6] While these impairments may be apparent in some individuals post-stroke, they may not be prevalent in community-ambulating stroke survivors. Orthopedic practitioners are likely to encounter and report on the most severely involved patients who require surgical correction of musculoskeletal deformities. Whether these stroke survivors are capable of independent community ambulation is not well described in available publications, but it seems unlikely given the seriousness of the musculoskeletal problems described.

Other studies have found decreased dorsiflexion PROM and increased plantar flexor muscle stiffness in stroke survivors with less readily apparent musculoskeletal impairments, some of whom were capable of walking.[4,5,10–14] However, these studies were designed to examine the effectiveness of exercise interventions for improving lower extremity PROM[11,14] or to identify the mechanisms contributing to musculoskeletal impairments post-stroke.[4,5,10,12,13] Hence, volunteers were selected on the basis of clinically evident contractures, spasticity, or increased muscle stiffness. To our knowledge, only two studies have examined passive musculoskeletal properties of the lower extremities in ambulatory stroke survivors selected solely on the basis

of locomotor status. Both studies demonstrated abnormally increased passive stiffness of the plantar flexors.[13,15] However, muscles and joints proximal to the ankle were not examined.

While the physiologic mechanisms and predisposing factors that lead to PROM deficits post-stroke are not fully understood, contributing factors include paresis, hyperreflexia, and muscle strength imbalance.[3,5,6,16] These impairments interfere with execution of voluntary motor commands and lead to disuse and immobilization of affected body parts.[1] When paretic muscles are immobilized in a shortened position, they adapt to their resting length and lose sarcomeres until those remaining overlap optimally to enable the muscle to develop maximal tension at the immobilized length.[17] This process results in a shortened end-to-end length of the affected muscle. Because poststroke hemiparesis results in immediate immobilization of affected muscles, this process may begin as early as the acute phase of the neural insult. For example, in the mouse soleus muscle, a 60% decrease in muscle fiber length has been observed after only 24 hours of immobilization.[18–21] In the presence of chronic immobilization, accumulation of intramuscular connective tissue, increased intramuscular fat, and degenerative changes in the myotendinous junction further contribute to decreased muscle length and increased muscle stiffness.[1]

Other poststroke neural impairments such as hyperreflexia and strength imbalances interact with immobilization to cause additional muscle shortening and to exacerbate soft-tissue changes.[22] Moreover, decreased extensibility of muscle makes any pulling force transmitted more readily to muscle spindles. Consequently, there is an increased spindle response to stretch that leads to more muscle shortening.[23,24]

It is unclear whether individuals with stroke who regain independent community ambulation have decreased lower extremity PROM. Standing and walking may counteract the effects of acute immobilization and help maintain normal muscle length and stiffness. However, locomotion alone may be inadequate to prevent muscular changes that lead to PROM deficits. As previously discussed, changes in muscle properties are caused not only by decreased mobility but also by hyperreflexia and muscle strength imbalances. These neuromuscular impairments are often observed regardless of

ambulatory status. For example, the clinical presentation of ambulatory and nonambulatory stroke survivors may include hyperactive quadriceps and Achilles tendon reflexes as well as weak dorsiflexors. These impairments, which encourage sustained knee extension and ankle plantar flexion, may contribute to changes in muscle properties and PROM. This theoretical argument is supported by the observations of Dietz and Berger[15] and Lamontagne et al,[13] who have shown increased ankle plantar stiffness in ambulatory stroke survivors.

The purpose of this study was twofold. First, we aimed to determine whether community-ambulating stroke survivors display decreased lower extremity PROM in paretic and nonparetic limbs. Second, we sought to determine whether PROM changes, if present, were caused by decreased length or increased stiffness of the muscles surrounding the joint. We hypothesized that, compared to able-bodied individuals, community ambulators with chronic poststroke hemiparesis would show decreased lower extremity PROM on the paretic but not the nonparetic side and that decreased PROM would be associated with increased muscle stiffness and decreased muscle length. Understanding whether lower extremity PROM is decreased in chronic stroke survivors who regain independent community ambulation may help physical therapists determine whether their patients are at risk of this musculoskeletal impairment. Consequently, clinicians can develop treatment programs that are most likely to address acute and chronic stroke-related impairments.

Methods

Subjects

To be included in the study, individuals with stroke had to have sustained a single, unilateral cortical or subcortical stroke at least six months before testing as indicated by diagnostic imaging reports in the medical record. Because we were interested in examining changes in lower extremity PROM that are associated with successful hemiparetic gait, participants had to be independent community ambulators who were able to work, complete activities of daily living (ie, shop, drive, catch a bus), or perform leisure activities (ie, go to the gym, a

restaurant, the movies) outside their home without the use of a wheelchair. The use of an assistive device was not exclusionary. Information about locomotor ability was obtained through self-report. The ability to ambulate independently in the community was further assessed by examiners' observations as to whether each participant was able to ambulate independently from the lobby of our building to the research laboratory, which involved walking 92 feet on carpeted and tiled surfaces, opening two doors, and negotiating an elevator. To ensure that we were examining a cohort of stroke survivors with residual poststroke impairment, subjects with stroke were included only if they exhibited clinical signs consistent with upper motor neuron syndrome such as hemiparesis, muscle strength imbalance, abnormal synergy patterns, impaired isolated joint movement, and hyperreflexia. The presence of upper neuron signs was confirmed in a brief physical examination conducted by one of the examiners (S.S.-I.), who is a licensed physical therapist. To be included in the study, able-bodied subjects had to show no signs of neurological disease and report no significant medical history of neurological disease or injury. Able-bodied and stroke subjects were excluded if they reported a significant medical history of any bone or joint pathology that could affect lower extremity PROM, such as joint replacement, arthritis, internal fixation, and recent fracture. Subjects were not excluded if physical examination revealed joint contracture, increased K, or decreased PROM that could not be attributed to bone or joint disease. To be included in the study, all volunteers had to be at least 21 years of age and able to provide informed consent.

Stroke subjects were recruited from a database of stroke survivors that is maintained at Northwestern University and from signs posted at a nearby gym that specializes in exercise programs for people with disabilities. Able-bodied participants were recruited from the Northwestern University faculty and staff as well as the general community by way of flyers posted in public areas near the laboratory.

Seventeen individuals with chronic poststroke hemiparesis (11 male, six female) and 15 able-bodied individuals (six male, nine female) who met the aforementioned inclusion criteria participated in the study. The mean (standard deviation) age of paretic and neurologically intact subjects was 58.7 (9.0) and 51.9 (14.5) years, respectively, which was not significantly different between groups

(independent *t* test, $P = 0.121$). One stroke subject had received an injection of botulinum toxin in the paretic gastrocnemii for spasticity management. This individual was included in the study, as we set no exclusion criteria a priori addressing this or other forms of medication. On average, subjects had sustained their stroke 6.3 (4.5) years before participating in this study, and all subjects were at least one year post-stroke. There were 13 subjects with left hemiparesis and four subjects with right hemiparesis. All hemiparetic volunteers used walking as their primary mode of ambulation at home and in the community and were able to walk independently with or without an assistive device at least 92 feet. Despite this level of function, those with stroke did not display normal walking ability. Gait impairments that are consistent with poststroke hemiparesis, such as foot drop, stiff-legged gait, and knee hyperextension, were evident on visual inspection of overground walking. Moreover, participants reported that they were unable to walk as well as before their stroke. For instance, some stroke survivors reported anecdotally that they were unable to walk quickly or run, and others indicated that their gait felt clumsy and uncoordinated. All subjects participated voluntarily and gave informed consent according to the Declaration of Helsinki and as approved by the Institutional Review Board at Northwestern University.

In this study, we were interested in examining changes in lower extremity PROM that are caused by changes in passive mechanical properties of muscle. When PROM is tested with biarticular muscles such as the rectus femoris, tensor fascia latae, hamstrings, and gastrocnemii lengthened across both joints that they cross, the maximum joint position that is achieved is largely a function of the length and stiffness of these muscles.[25] Hence, we tested hip extension with knee flexion to examine rectus femoris and tensor fascia latae, hip flexion with knee extension to examine hamstrings, and ankle dorsiflexion with the knee flexed 30 degrees to examine gastrocnemii. For each of the three joint movements examined, three dependent variables were measured: the maximum joint angle achieved by passively rotating the limb (ANG_{max}), the torque required to achieve the maximum joint angle (T_{max}), and the passive K, which is the change in torque per unit of change in joint angle and represents the amount of torque required to rotate the joint one degree. These measurements provide insight into lower extremity PROM as well as the mechanical properties of the muscles surrounding the joint. As

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further indicated in the Discussion section, they also shed light on the muscle properties underlying between-group differences in PROM. The following sections describe the test procedures in detail.

Instrumentation

As depicted in Figure 1, a Biodex dynamometer (Biodex Medical Systems, Shirley, NY) was used to passively rotate the hip and ankle at a constant velocity of 5 degrees/sec while measuring the angular joint position and net joint torque to an accuracy of ±1 degree and ±7 Nm, respectively. We reasoned that this movement velocity would be slow enough to prevent stretch reflex excitation of the muscles being lengthened. The reliability and validity of position, torque, and velocity measures obtained from the Biodex dynamometer have been demonstrated previously.[26–28] In short, if care was taken to align the axis of rotation of the joint with the dynamometer axis of rotation and if movement speeds do not exceed 300 degrees/sec, the Biodex provides valid and reliable measurements of joint position, torque, and velocity.

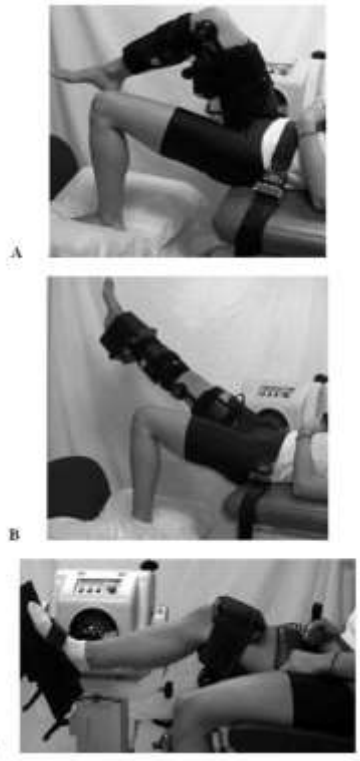

FIGURE 1 Experimental setup for passive range of motion testing. A. Hip extension. B. Hip flexion. C. Ankle dorsiflexion.

Bipolar silver surface electrodes (DelSys, Inc., 10 mm length, one mm width, one cm interelectrode distance) were used to monitor electromyography (EMG) activity from the medial gastrocnemius, semimembranosus, and rectus femoris during passive limb movement to ensure that muscle activity remained quiet during the stretching procedure. EMG signals were amplified 10 times at the electrode site before remote differential amplification (common mode rejection ratio, 92 dB; gain range, 100–10,000 times; frequency response, 20–450 Hz). In preparation for placement of the EMG electrodes, the skin over each muscle was cleaned and gently abraded with an alcohol swab. Surface EMG electrodes were placed over the distal half of the medial gastrocnemius, semimembranosus, and rectus femoris muscles of both legs in neurologically intact and stroke subjects. A common reference electrode was placed over each tibia on the anterior aspect of the leg. Electrodes were secured with adhesive tape to prevent electrode movement during the experiment.

Protocol

Subjects were positioned for PROM testing as shown in Figure 1. During the hip tests, subjects were positioned supine on a firm examining table next to the dynamometer (Fig. 1A and B). During the ankle tests, subjects were seated in the chair of the Biodex system (Fig. 1C). The test limb was secured to the arm of the Biodex dynamometer in each of the three test configurations described below. In each configuration, the joint axis of rotation (either the hip or the ankle) was aligned with the axis of rotation of the dynamometer. Before testing, one of the experimenters manually rotated the test joint to its maximum angular position, which was defined as the joint position at which the pelvis began to rotate during the hip tests or the joint position at which the heel began to lose contact with the Biodex footplate for the ankle test. The maximum angular position of the joint was determined through palpation and visual inspection. Software "stops" were set in the Biodex controller at the joint angles defining the start and the end of joint PROM, and, henceforth, the joint was rotated through this PROM. During testing, the dynamometer rotated the joint of interest through this preset PROM three times. Each test

was completed bilaterally on each volunteer. For safety purposes, mechanical stops were positioned approximately five degrees beyond the software stops. Subjects were instructed to relax completely and allow the dynamometer to passively rotate their limb. Surface EMG was monitored on an oscilloscope for bursts of activity that were greater than the EMG activity observed before joint rotation was initiated. If EMG activity increased to three times that observed before joint movement, the subject was reminded to relax, and the test was restarted. With minimal cueing, all subjects were able to remain relaxed during testing, and some subjects fell asleep. The right leg of control subjects and the nonparetic limb of stroke survivors were always tested first. The order in which each motion was examined was counterbalanced to avoid an ordering effect.

Hip Extension Test

To examine hip extension PROM, we passively extended the hip while the knee was positioned at 90 degrees of flexion using a knee brace shown in Figure 1A. In this position, the rectus femoris and tensor fascia latae were lengthened across the hip and the knee joints.[29] Subjects were positioned supine with their pelvis in neutral and firmly secured to the examination table with a wide, nonextensible nylon strap that buckled and cinched tight like a seat belt. Both ischial tuberosities hung slightly off the edge of the examining table. The test leg was secured to the dynamometer arm with a Velcro strap wrapped snuggly around the mid thigh. The leg that was not being tested was placed on a platform at the end of the plinth. For all subjects, the starting joint angle for this test was 60 degrees of hip flexion (defined as −60 degrees in subsequent figures). The maximum angular position for hip extension was defined as the hip position at which the pelvis began to tilt anteriorly, as evidenced by visible or palpable movement of the anterior superior iliac spine.

Hip Flexion Test

To examine hip flexion PROM, we passively flexed the hip while the knee was fully extended, as shown in Figure 1B. This test resembled the straight leg raise test for hamstring length in which the hamstrings are lengthened across the hip and the knee joint.[29]

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Subjects were positioned supine with their pelvis in neutral and firmly secured to the examination table. Both ischial tuberosities hung slightly off the edge of the examining table. The knee of the test leg was maintained in full extension with a knee brace. The test leg was secured to the dynamometer arm with a Velcro strap wrapped snuggly around the distal tibia. The leg that was not being tested was placed on a platform at the end of the plinth. For all subjects, the starting joint angle for this test was zero degrees of hip flexion. The maximum joint angle was defined as the hip position at which the pelvis began to tilt posteriorly, as evidenced by visible or palpable movement of the anterior superior iliac spine.

Ankle Dorsiflexion Test

Ankle dorsiflexion PROM was examined by passively dorsiflexing the ankle with the knee flexed 30 degrees (Fig. 1C). Technical limitations prevented us from fully extending the knee. Subjects were seated with the knee of the test leg supported by a padded bolster. The foot was pressed firmly against a foot plate that was attached to the dynamometer arm. A Velcro strap was used to secure the foot to the foot plate. For all subjects, the starting joint angle for this test was 30 degrees of plantar flexion (defined as −30 degrees in subsequent figures). The maximum angular position of the joint was defined as the ankle angle at which the heel began to lose contact with the foot plate.

Data Processing and Analysis

Angular position and torque data measured from the Biodex system were sampled online at 1000 Hz via a 12-bit analog to digital converter and Labview software (National Instruments). We were interested in the torque generated by passive lengthening of muscle as a function of joint angle. However, the torque transducer in the Biodex system measured the total torque applied to the dynamometer, which includes the torque generated by passive tissue stretch as well as torque due to the effect of gravity acting on the limb. Therefore, it was necessary to adjust the torque measurements for gravity. Because the torque caused by gravity varies as a function of joint angle, the following equation was employed: $T_s = T_t - (T_g \cos\theta)$, where T_s = torque due to passive tissue stretch, T_t = total torque measured at the

transducer, T_g = torque due to the effect of gravity on the limb, when the limb is parallel to the floor, and θ = joint angle, measured with respect to the floor.

To calculate T_g, torque was measured immediately before testing while the limb rested in the start position and the subject was instructed to relax. T_s was considered to be zero in the start position. Therefore, T_g was calculated from this initial measurement as follows: $T_g = T_t/\cos\theta$.

Calculations were performed online in Labview software, after which position and T_s data were down-sampled to 10 Hz and saved to a personal computer.

Each of the dependent variables (ANG_{max}, T_{max}, and K) was calculated in Matlab after the torque and position data were low pass filtered (15th order, zero lag Butterworth, 0.75-Hz cutoff frequency). Mean passive K was calculated by plotting T_s against the joint angle across the entire PROM for each of the three movement repetitions and then calculating an ensemble average of the three curves (Fig. 2). The relationship between T_s and joint angle was fit with a second-order polynomial function, as the data were well described by second-order fit ($R^2 > 0.90$ for all three joint movements). K was calculated by differentiating the best fit curves and then calculating the mean derivative across the movement cycle. When this second-order polynomial function was differentiated, the output was the rate of change of torque (ie, stiffness) at each point in the movement cycle. By calculating the mean derivative, we obtained the average rate of change of torque across the movement cycle (K). A representative example of these data is shown in Figure 2.

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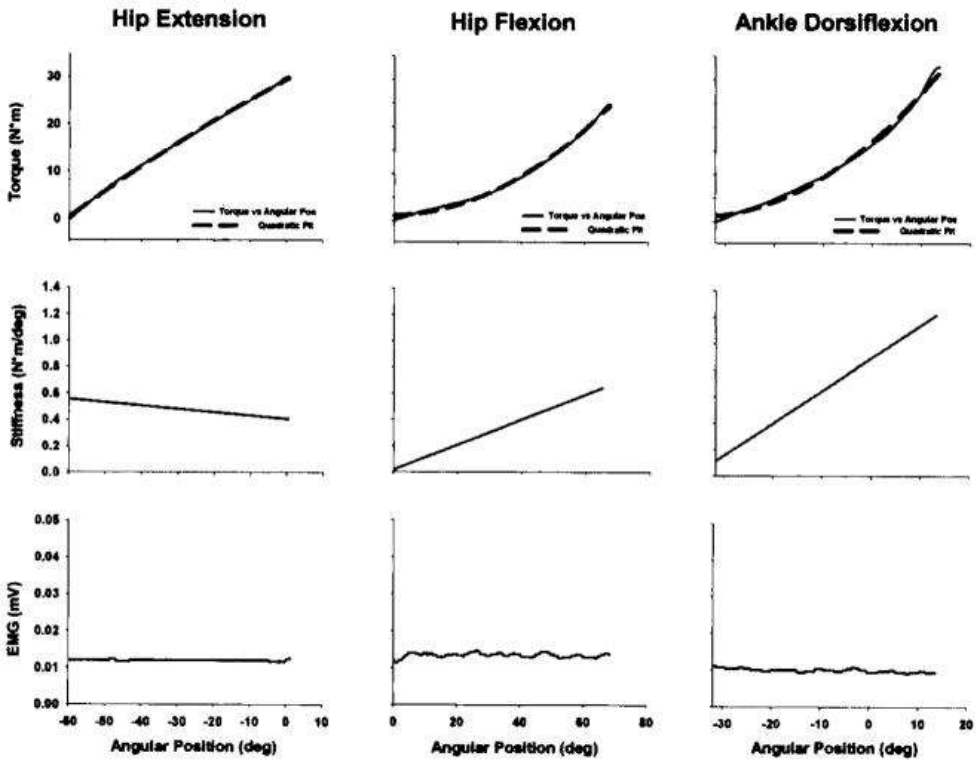

FIGURE 2 Representative data from a single control subject. Top. Torque versus angular joint position (solid line) fit with a quadratic curve (dashed line) across the entire passive range of motion (PROM) for hip extension, hip flexion, and ankle dorsiflexion. Middle. Passive joint stiffness (K) calculated by differentiating the quadratic fit of the torque versus angular joint position curve and then calculating the derivative across the entire PROM. Bottom. Rectified electromyography (EMG) activity recorded from the rectus femoris, semimembranosis, and medial gastrocnemius, respectively, during the hip extension, hip flexion, and ankle dorsiflexion movement cycles.

Values for ANG_{max} and T_{max} were calculated for each of the three movement repetitions, but no meaningful differences among repetitions were observed. Hence, the mean of the three repetitions was used in group analysis. Data from the left and right limbs of each able-bodied subject was averaged to form a single control group for each dependent variable. The Kolmogorov-Smirnov test was used to test for normality in the data. Significant deviations from normality were seen for several dependent variables. Hence, the non-parametric statistics described below were applied.

Between-group differences among the three groups (able-bodied versus paretic versus nonparetic) were examined using

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Kruskal-Wallis analysis of variance. In the presence of a significant main effect, the minimum significant difference (MSD) test[30] was used to identify significant differences between able-bodied and paretic and able-bodied and nonparetic values. The MSD test is a multiple comparison procedure that controls for type I error.[30] All statistical tests were done in SPSS (SPSS, Inc., Chicago, IL) except for the MSD test, which was calculated by hand using the ranks obtained from the Kruskal-Wallis test. Differences were considered significant at $P \leq 0.05$.

Results

Group mean (standard error) and the range of values for each dependent variable are visually depicted in Figures 3 and and44 and numerically presented in Table 1.

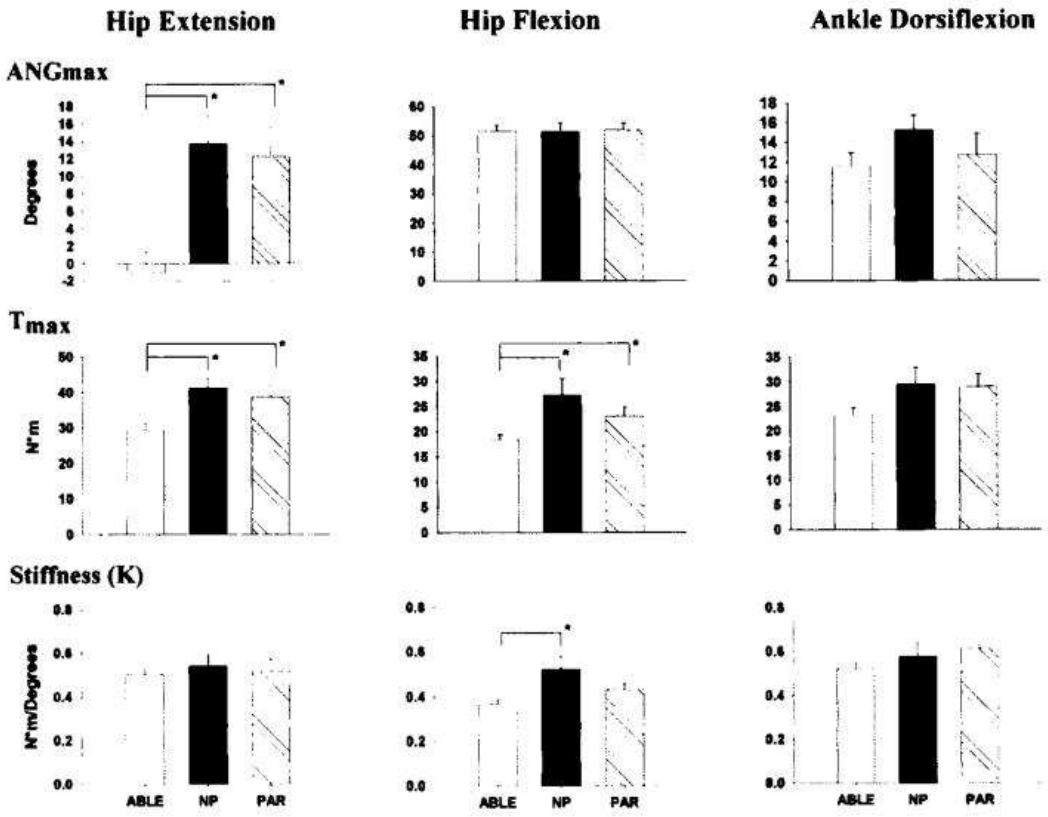

FIGURE 3 Group data (mean and standard error) for able-bodied, nonparetic, and paretic limbs. Top. Maximum angular position of the joint (ANG_{max}) for hip extension, hip flexion, and ankle dorsiflexion. Middle. Torque required to reach maximum position

of the joint (T_{max}). Bottom: Mean passive joint stiffness (K) for each joint movement. Able, able-bodied; NP, nonparetic; PAR, paretic.

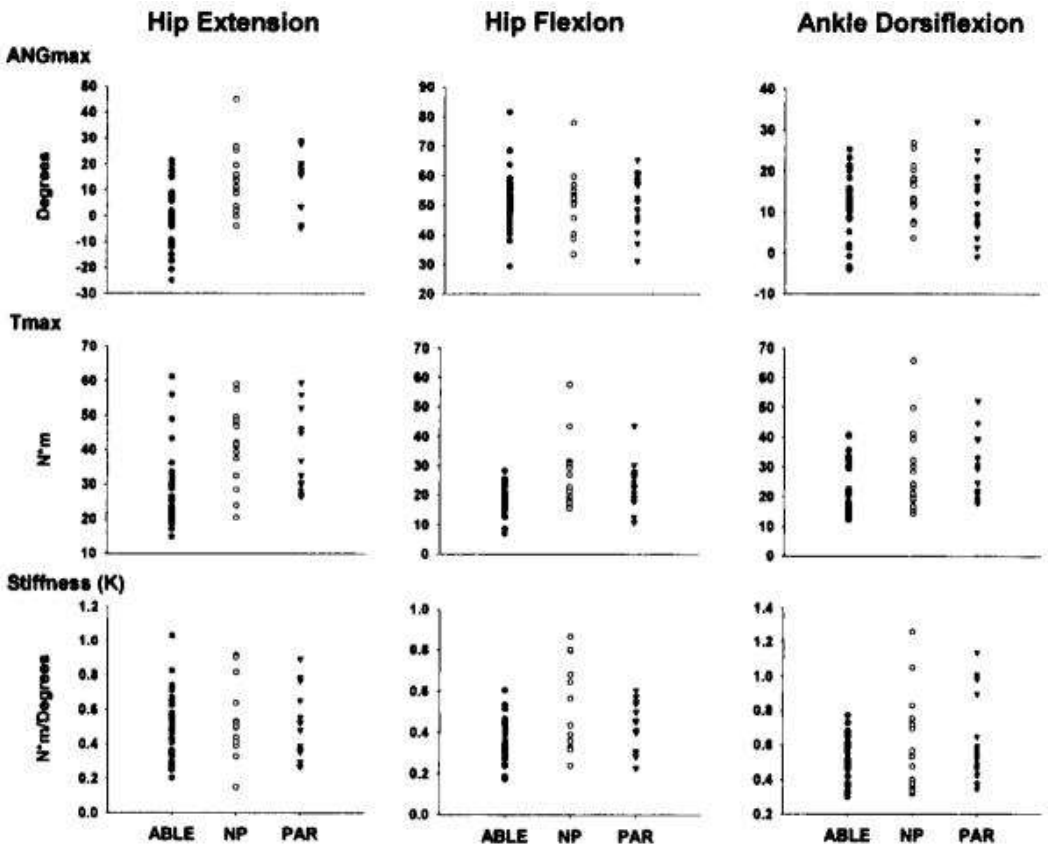

FIGURE 4 Data from each participant showing the range of values observed. The organization of the figure and abbreviations are the same as in Figure 3.

TABLE 1 Group Means (Standard Error) and Range of Values for ANG_{max} T_{max} and Joint Stiffness (K) Observed Within Each Group for Each PROM Test

	Able-bodied	**Nonparetic**	**Paretic**
Hip extension			
ANG_{max}, mean (SE)	−1.05 (2.352)	13.69 (3.17)[a]	12.27 (3.29)[a]
Range	Min: −24.90, max: 21.53	Min: −3.71, max: 45.07	Min: −4.57, max: 29.10
T_{max}, mean (SE)	29.27 (2.0)	41.12 (2.9)[a]	38.75 (3.22)[a]
Range	Min: 14.88, max: 61.26	Min: 20.48, max: 59.07	Min: 26.43, max: 59.51
Joint stiffness (K), mean (SE)	0.50 (0.04)	0.54 (0.05)	0.52 (0.06)
Range	Min: 0.20, max: 1.03	Min: 0.15, max: 0.92	Min: 0.27, max: 0.89
Hip flexion			
ANG_{max}, mean (SE)	51.61 (1.89)	51.39 (3.06)	51.98 (2.29)
Range	Min: 29.51, max: 81.52	Min: 33.56, max: 78.04	Min: 31.45, max: 65.42
T_{max}, mean (SE)	18.42 (0.88)	27.17 (3.33)[a]	23.10 (1.81)[a]
Range	Min: 7.00, max: 28.24	Min: 15.68, max: 57.65	Min: 10.95, max: 43.77
Joint stiffness (K), mean (SE)	0.37 (0.02)	0.52 (0.06)[a]	0.43 (0.03)
Range	Min: 0.17, max: 0.61	Min: 0.24, max: 0.87	Min: 0.23, max: 0.61
Ankle dorsiflexion			
ANG_{max}, mean (SE)	11.55 (1.4)	15.20 (1.52)	12.78 (2.13)
Range	Min: −3.97, max: 25.27	Min: 3.62, max: 26.91	Min: −0.83, max: 32.10
T_{max}, mean (SE)	23.17 (1.66)	29.46 (3.38)	29.08 (2.40)
Range	Min: 12.33, max: 40.76	Min: 14.37, max: 65.78	Min: 17.97, max: 52.21
Joint stiffness (K), mean (SE)	0.52 (0.03)	0.57 (0.07)	0.61 (0.06)
Range	Min: 0.30, max: 0.77	Min: 0.32, max: 1.26	Min: 0.35, max: 1.14

[a]Indicates that values were significantly different from control values, at $P \leq 0.05$ per the minimum significant difference test.

Abbreviations: ANG_{max} maximum angular position of the joint; SE, standard error; T_{max} torque required to achieve ANG_{max}.

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Across all three joint movements examined, between-group differences in ANG_{max} were observed only during hip extension where ANG_{max} was higher in the paretic and nonparetic limbs of poststroke subjects compared to able-bodied subjects ($P \leq 0.05$; MSD). Note that negative values indicate that the hip did not extend beyond a flexed posture at ANG_{max}. Hip flexion and ankle dorsiflexion ANG_{max} were not different among groups (hip flexion: $P = 0.79$, dorsiflexion: $P = 0.31$, Kruskal-Wallis).

T_{max} during hip extension was significantly higher in paretic and nonparetic limbs of stroke survivors compared to control limbs ($P \leq 0.05$; MSD). Compared to the able-bodied group, significantly more torque was also required to reach ANG_{max} during hip flexion in paretic and nonparetic limbs of poststroke subjects ($P \leq 0.05$; MSD). There was no significant difference among groups in T_{max} measured during passive ankle dorsiflexion ($P = 0.07$, Kruskal-Wallis).

Passive K measured during hip extension was not different among groups ($P = 0.80$, Kruskal-Wallis). K during hip flexion was higher in the nonparetic ($P \leq 0.05$; MSD test) but not the paretic limb of poststroke subjects. There was no significant difference among groups in K measured during passive ankle dorsiflexion ($P = 0.63$, Kruskal-Wallis).

Discussion

We initially hypothesized that community-ambulating stroke survivors with residual stroke-related impairment would show decreased PROM in their paretic lower limbs that would be associated with increased muscle stiffness and decreased muscle length. The data that we present here fail to support this hypothesis, as the cohort of stroke survivors examined did not display decreased passive hip extension, hip flexion, or ankle dorsiflexion in paretic or nonparetic limbs. Passive hip extension in both lower limbs of stroke survivors was greater than that observed in the able-bodied group, and passive ankle dorsiflexion and hip flexion were not different among groups. Moreover, increased passive K was not observed in the paretic limb during any motion examined. Increased passive K was seen only during hip flexion in the nonparetic limb of stroke survivors.

In this study, we were interested in identifying changes in lower extremity PROM and that could be attributed to changes in muscle properties. Hence, our measurements were made with biarticular muscles such as the hamstrings, rectus femoris, and tensor fasciae latae lengthened across both joints that they cross. The gastrocnemii were not fully lengthened at the knee because of technical limitations in our experimental setup that prevented us from extending the knee beyond 30 degrees of flexion. However, by stabilizing the knee and preventing knee flexion, we were able to lengthen the gastrocnemii by dorsiflexing the ankle. In these limb configurations, ANG_{max} was determined primarily by the passive length and stiffness of two-joint muscles.[29] Therefore, our data suggest that lower extremity biarticular passive muscle length is not decreased and that passive muscle stiffness is not increased in the paretic lower limbs of the community-ambulating stroke survivors examined here.

Our observations differ from previous reports indicating that paretic lower extremity PROM is decreased and that passive K is increased post-stroke. Differences between our results and those of previous studies are particularly evident at the ankle where others have found ankle plantar flexion contractures as great as 20 degrees and equinus deformities.[3,7,13,31,32] Moreover, others have shown that paretic plantar flexion stiffness is up to 10 times greater in paretic compared to control limbs.[10,12,33] Previous reports have also shown substantial loss of hamstring muscle length as well as hip and toe flexion contractures that interfere with positioning and mobility.[3,6]

Differences in the level of neuromuscular impairment and locomotor ability between the subjects examined here and those described in previous studies are the most plausible explanations for these disparities. For example, some review papers[2,3] and research reports[6,7] suggest that hip flexion, knee flexion, and ankle plantar flexion contractures are among the most common deformities observed in stroke survivors and that these problems are caused by muscle changes. However, many of these observations come from the orthopedic surgery literature in which clinicians and researchers examine PROM deficits before and after surgical intervention. While the ambulatory status of these individuals is not reported in the literature, it seems unlikely that these surgical candidates would

possess ambulation skills that are as sophisticated as those observed in the stroke survivors whom we tested.

Other studies reported in the literature have been designed to examine the effectiveness of nonsurgical interventions[11,14,31] for improving lower extremity PROM or to identify the mechanisms contributing to musculoskeletal impairments post-stroke.[4,5,10,12,13] These studies typically show that ankle plantar flexion PROM is decreased approximately 50% and that stiffness or resistance torque can be increased 1.5 times in paretic limbs of stroke survivors. However, in these studies, volunteers were selected based on the clinical presence of calf muscle stiffness, increased Ashworth scores, and/or demonstrable ankle plantar flexion contractures. In the present study, we had no a priori knowledge of the integrity of subjects' neuromuscular system. Rather, stroke subjects were selected if they reported the ability to ambulate independently in the community. Therefore, unlike the aforementioned studies, our inclusion criteria did not favor the selection of individuals with increased muscle stiffness. Consequently, we may have examined a cohort of stroke survivors with less hyperreflexia, muscle imbalance, or other impairments likely to contribute to changes in passive muscle properties.

Other studies have examined passive muscle stiffness in stroke survivors specifically selected for their locomotor ability.[13,15] These studies have shown that intrinsic muscle stiffness is increased during poststroke gait. For example, Dietz and Berger[15] showed that tension develops in hemiparetic calf muscles during the stance phase of gait without a concomitant increase in muscle activity. Lamontagne et al[13] showed that the relative contribution of the passive component to total plantar flexion torque during gait was increased in paretic limbs of stroke survivors compared to nonparetic and control limbs. However, increased passive tension in paretic muscles occurred only in individuals who could not produce adequate active tension. Consequently, the more severely hemiparetic individuals were most likely to exhibit high passive muscle stiffness. These data suggest that abnormally high levels of passive muscle stiffness may compensate for inadequate active muscle tension. While a limited description of the locomotor ability of subjects examined in the previous studies prevents comparison with our sample, it is possible that those individuals with

increased passive muscle stiffness had less well recovered walking ability than the group examined here.

Alternatively, we may have failed to detect muscle stiffness changes because we tested people at rest using a slow (5 degrees/sec) movement velocity during nonfunctional movements. Others have shown that muscles have viscosity behaviors whereby resistance to passive movements increases with movement velocity.[33] If we had examined PROM and K at faster movement velocities or during a functional task, we may have observed the stiffness changes that have been reported previously.

One unexpected finding of this study was that subjects with stroke had more passive hip extension than those without stroke. In the stroke group, passive hip extension was approximately 13 degrees and did not differ between the paretic and nonparetic sides. In contrast, hip extension in the able-bodied group was approximately −1 degree bilaterally. Similarly, there was a statistically insignificant tendency for ankle dorsiflexion PROM to be greater in the stroke group compared to the control group. While these observations appear to suggest that rectus femoris, tensor fasciae latae, and perhaps gastrocnemius muscle length were increased above normal values in the community-ambulating stroke survivors examined here, this explanation may not be accurate. Between-group differences in hip extension and dorsiflexion PROM may be partly related to PROM deficits in the able-bodied group. According to Kendall and colleagues,[29] the normative value for hip extension with knee flexion is 10 degrees. Other investigators have reported 15 to 20 degrees of ankle dorsiflexion in control subjects tested under similar conditions.[11,12] During our hip extension test, less than 15% of able-bodied limbs achieved 10 degrees or more of hip extension, while 64% of stroke limbs reached the normative value. Moreover, our control subjects only reached approximately 12 degrees of dorsiflexion. These observations may be consistent with those of Kendall et al[29] and Sahrmann[34] who suggest that muscle length deficits are prevalent in the general population.

A second seemingly counterintuitive finding was that paretic and nonparetic T_{max} was increased above control values during the hip extension test. Higher values for T_{max} could be indicative of increased

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muscle stiffness in the biarticular hip flexors. However, passive K was not abnormally increased during the same test. Therefore, it is more likely that increased values for T_{max} are indicative of increased extensibility of the rectus femoris and tensor fascia latae in the stroke group compared to the able-bodied group. When skeletal muscle is passively stretched, it generates a restoring force that is proportional to the magnitude of the stretch.[35] Hence, higher levels of T_{max} during hip extension in the stroke group are expected in light of the fact that maximum hip extension was also increased in this group.

The question remains as to why hip extension (and perhaps ankle dorsiflexion) PROM was greater in the stroke group compared to the able-bodied group. This observation is counter to our theoretical framework that suggests that hyperreflexia, muscle imbalance, and paresis should lead to decreased PROM in stroke survivors. If we assume that our stroke group was similar to the control group before they had their stroke, then we must assume that the stroke group gained PROM after their stroke. Loss of muscle cross-sectional area is one possible explanation for these observations. Investigators such as Jorgensen and Jacobsen,[36] Pang et al,[37] and Scelsi et al[38] have demonstrated loss of lean (muscle) mass, muscle atrophy, and decreased muscle fiber diameter in paretic lower extremities of individuals with stroke. Hence, it is possible that decreased girth in the large muscles of the thigh resulted in more compliant tissue that could be more easily lengthened. Another possibility is that the mechanical characteristics of the trunk muscles may have been different in the stroke group compared to controls. In this study, hip extension ANG_{max} was defined as the joint position at which the pelvis began to rotate anteriorly. If the trunk or posterior hip muscles of stroke survivors were stiffer than normal, they may have been able to stabilize the pelvis against higher forces while the hip continued to extend. However, we did not examine trunk muscle stiffness or muscle girth in this study; so we are unable to make these assertions with certitude.

In contrast to our observations in hip extension, during poststroke hip flexion, appropriate values for ANG_{max} were accompanied by increased T_{max}. K was also increased during nonparetic hip flexion, and there was a trend toward increased K on the paretic side. These data are suggestive of increased passive stiffness in the poststroke hamstring muscles. In the presence of

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increased hamstring stiffness, higher than control levels of torque were required to achieve comparable values for ANG_{max}. The explanation for increased passive stiffness in the nonparetic hamstring is unclear, and our framework for understanding musculoskeletal adaptations post-stroke cannot easily explain these findings because nonparetic limbs do not display upper motor neuron signs. Consequently, while one might predict some increase in muscle stiffness on the nonparetic side due to an overall reduction in activity, one would also predict a larger increase in stiffness on the paretic side. However, this was not the case. These data suggest that factors other than poststroke neuromuscular impairment may contribute to musculoskeletal adaptations post-stroke. One possible explanation is use-dependent changes in muscle activation patterns that contribute to recovery of locomotion. Future studies will need to examine this possibility.

This study was limited in that it examined chronic stroke survivors who identified themselves as community ambulators based on their ability to work, complete activities of daily living, or perform leisure activities outside their home without a wheelchair. Thus, our results cannot be generalized to the entire population of stroke survivors. As indicated above, there are other (perhaps less well recovered) subpopulations of stroke survivors who have PROM problems. Another limitation of this study is the fact that we did not quantify the magnitude or extent of neuromuscular impairment in the stroke survivors examined. Participants were simply screened for the presence or absence of upper motor neuron signs such as hemiparesis, muscle strength imbalance, abnormal synergy patterns, impaired isolated joint movement, and hypertonicity and admitted into the study if there was at least one positive test. Hence, this study cannot examine the relationship between the extent of neuromuscular impairment and lower extremity PROM. A third limitation is the fact that we examined muscle length and stiffness during slow (5 degrees/sec) passive movements that were nonfunctional in nature. This design was selected in order to avoid eliciting stretch reflexes and to isolate the passive mechanical properties of muscle. However, many stroke survivors that we have encountered complain of an inability to run or walk quickly, and other investigators have reported a velocity-dependent increase in passive muscle stiffness post-stroke.[33] It remains possible that, at higher velocities of movement or during functional tasks, differences in passive K may emerge. Finally, it

remains possible that failure to see decreased ankle dorsiflexion PROM was due to the knee position used, which did not allow full elongation of the gastrocnemii at the knee. However, several other studies that report deficits in ankle dorsiflexion PROM have observed these results with the knee flexed 20 to 90 degrees.[10–12, 33,39] Consequently, future studies should examine stroke survivors with a broader range of walking abilities and neuromuscular impairments. A record of individual Fugl-Meyer scores and walking velocities would be helpful in ascertaining relationships among neuromuscular impairment, functional ability, and lower extremity PROM. Future studies should also consider examining lower extremity passive muscle properties during functional tasks, faster movements, and with biarticular muscles lengthened across both joints.

Conclusion

This study demonstrates that community-ambulating stroke survivors with residual upper motor neuron signs do not exhibit decreased lower extremity PROM caused by increased muscle stiffness or decrease muscle length. In fact, this subpopulation of stroke survivors appears to have more hip extension PROM than age-matched able-bodied individuals. This result was unexpected in light of the existing framework suggesting that poststroke hyperreflexia, muscle imbalance, and paresis are associated with decreased extensibility of skeletal muscle. The reason that lower extremity PROM is adequately maintained in this subpopulation of stroke survivors remains unclear, but may be related to the extent of neuromuscular recovery, activity level, exposure to exercise and rehabilitation, or use-dependent changes in muscle properties.

The clinical implications of these data are important and suggest that lower extremity PROM does not interfere with mobility in community-ambulating stroke survivors because the PROM values observed here are within those needed for gait.[40] While we cannot eliminate the possibility that stretching or other forms of exercise may have contributed to this outcome, it seems unlikely that additional stretching would improve locomotor ability in these individuals. Hence, if a physical therapist is certain that a stroke survivor is or will become a community ambulator, it may be prudent to place minimal emphasis

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on lower extremity stretching and to suggest other activities that may be more likely to address neuromuscular impairments. This finding is particularly important in light of recent evidence suggesting that physical therapists working with outpatient stroke survivors employ passive exercise in approximately 30% of sessions that address the lower extremities.[8]

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